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Analysis of Cardiac Physiology

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BM2102 – Modelling and Analysis of Physiological Systems

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Contents

1 Introduction

Introduction

This report presents a simulation-based analysis of cardiac physiology using the **CircAdapt Simulator v1.1.0**, focusing on normal sinus rhythm and progressive aortic valve stenosis (AS)—a pathological narrowing of the aortic valve that increases left ventricular workload.

In Part 1, cardiac pressures, flow velocities, and pressure-volume (PV) loops were analyzed under normal conditions and interpreted with reference to the *Wiggers diagram*, a standard representation of the cardiac cycle. In Part 2, the aortic valve area was progressively reduced up to 80% to simulate AS, and the resulting hemodynamic changes were evaluated.

The objective is to understand how AS alters cardiac performance, with particular attention to preload, afterload, stroke volume, and ventricular adaptation.

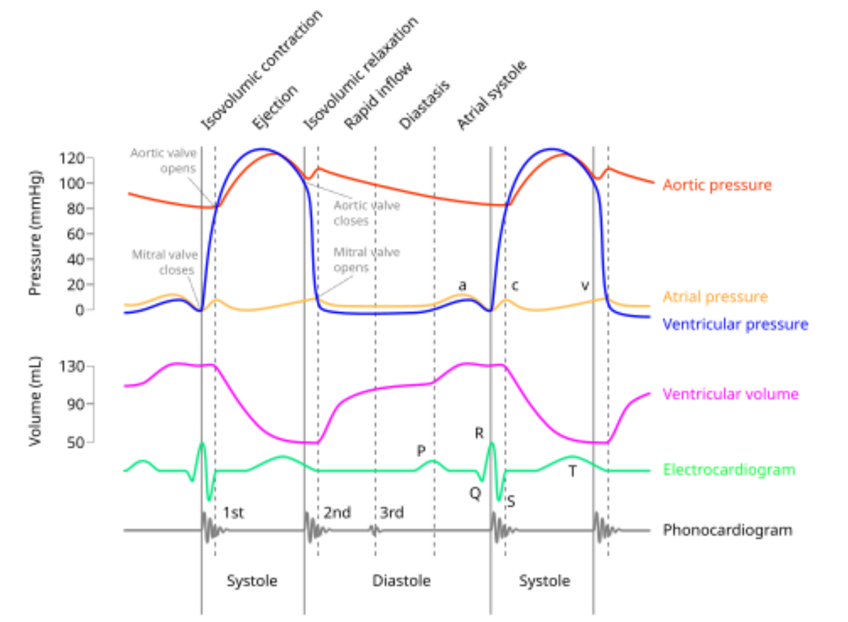


Figure 1: Wigger's diagram

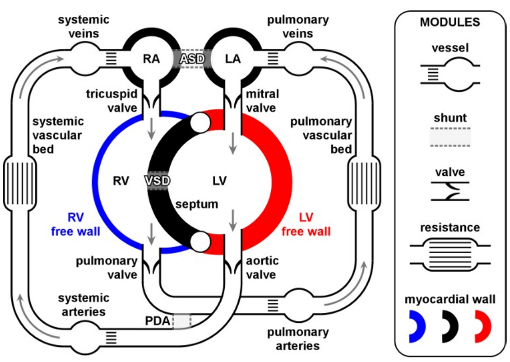


Figure 2: Model of the heart used by CircAdapt

a. Aortic Valve Opening and Closing

The left ventricular (LV) and aortic pressures were analyzed to determine the aortic valve's opening and closing times. The aortic valve opens when LV pressure exceeds aortic pressure (approximately 80 mmHg), marked by a vertical line in the reference column. It closes when LV pressure falls below aortic pressure. This corresponds to the ejection phase, where LV volume decreases from around 120 mL to around 50 mL.

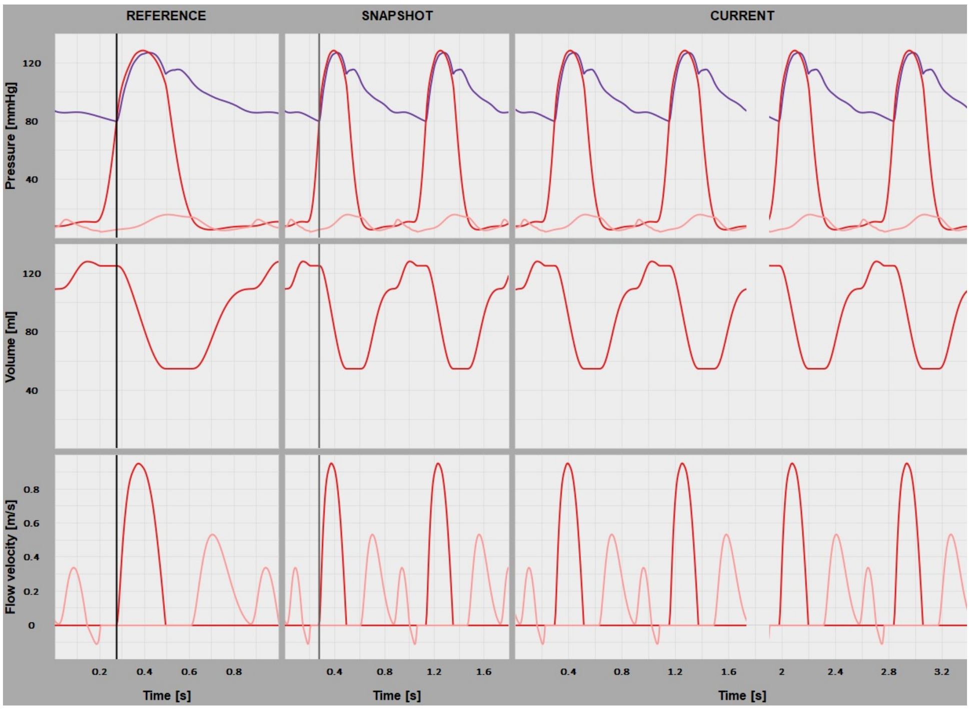


Figure 3: Screenshot of pressure and volume graphs showing aortic valve opening.

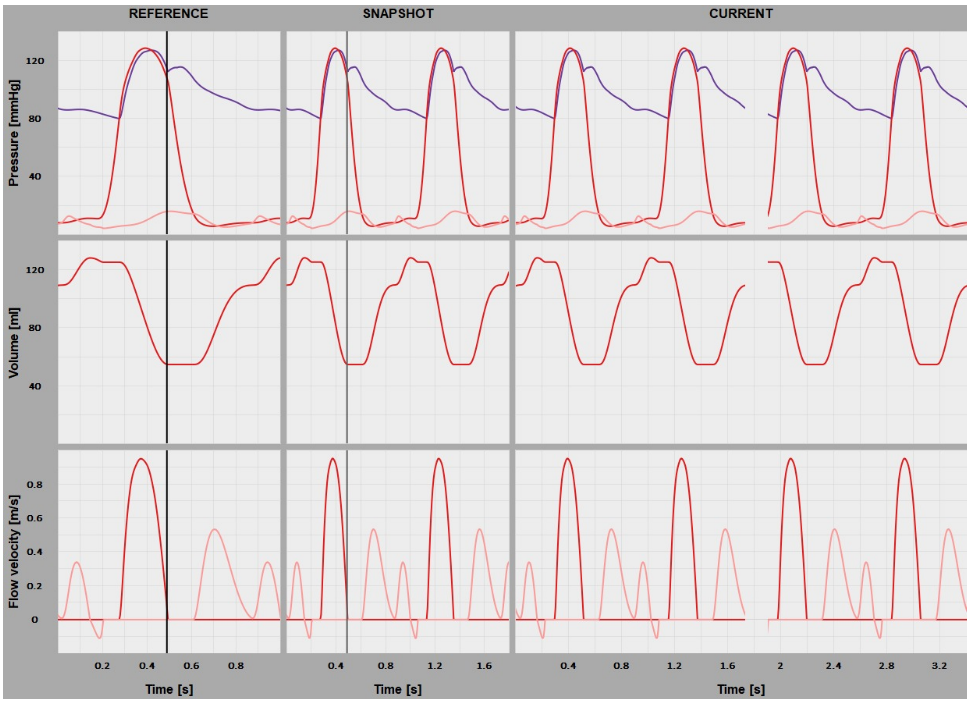


Figure 4: Screenshot of pressure and volume graphs showing aortic valve closing.

b. Mitral Valve Opening and Closing

The mitral valve opens when left atrial (LA) pressure exceeds LV pressure, initiating the filling phase. It closes when LV pressure rises above LA pressure. During the filling phase, LV volume increases from around 50 mL to around 120 mL.

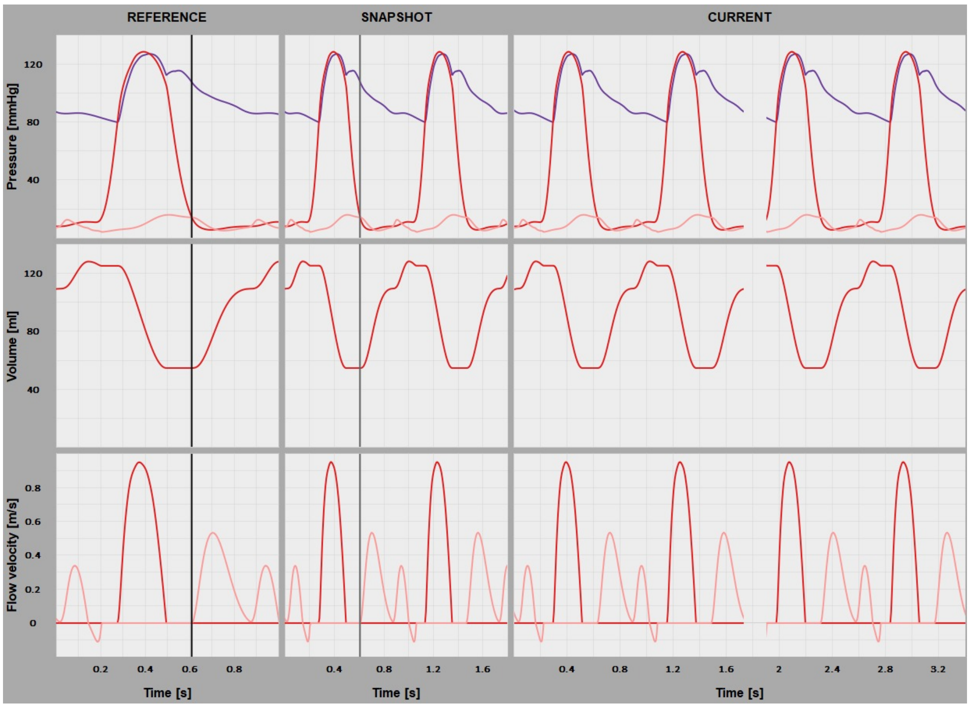


Figure 5: Screenshot of pressure and volume graphs showing mitral valve opening point.

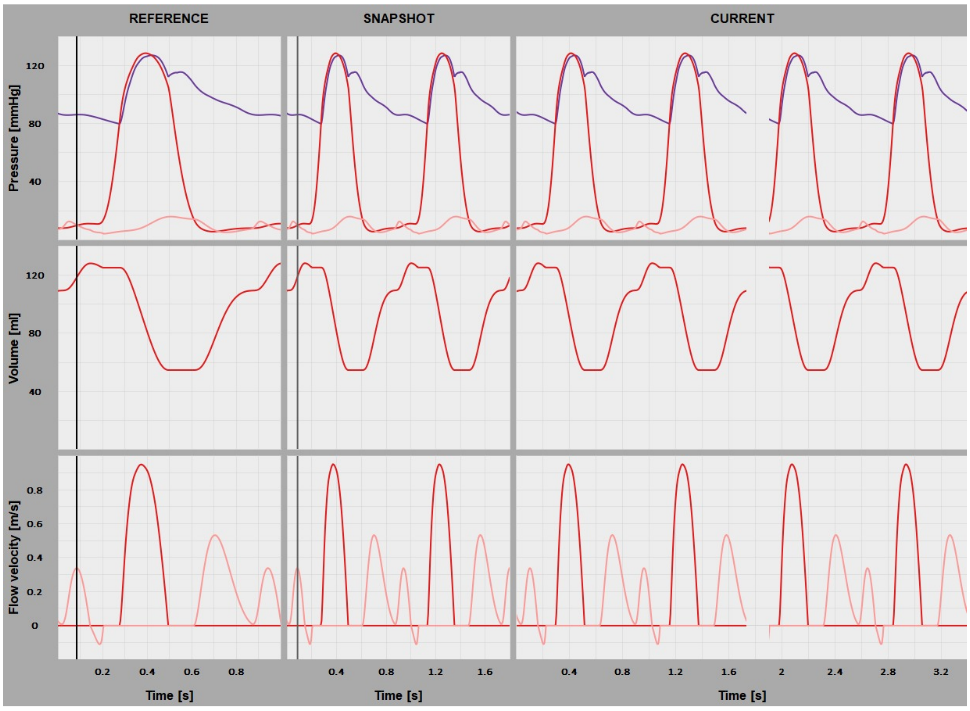
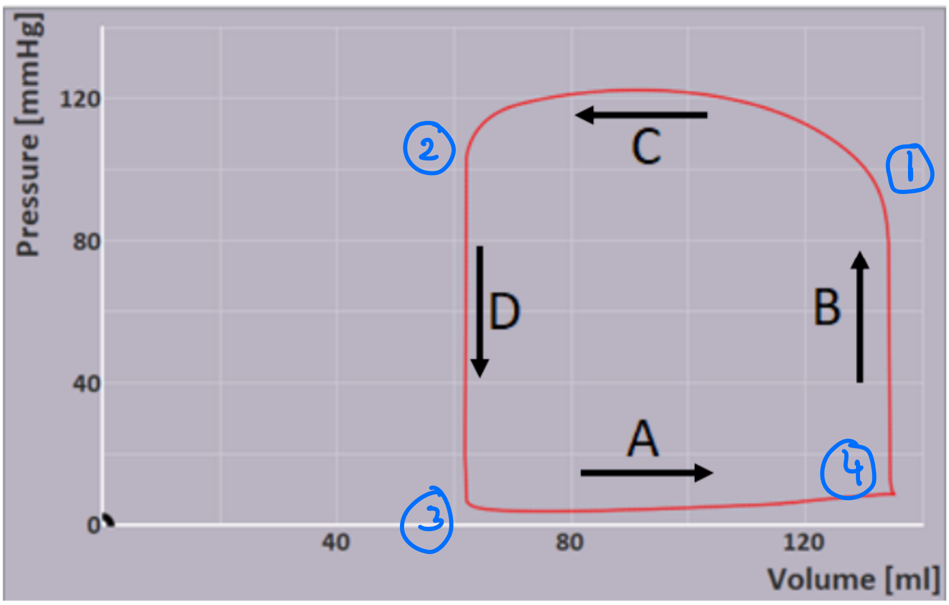


Figure 6: Screenshot of pressure and volume graphs showing mitral valve closing point.

c. Pressure-Volume Relation

The pressure-volume (PV) loop was labeled with points corresponding to valve events:

- 1: Aortic valve opening (high pressure, max volume).
- 2: Aortic valve closing (high pressure, min volume).
- 3: Mitral valve opening (low pressure, min volume).
- 4: Mitral valve closing (low pressure, max volume).



d. Cardiac Cycle Phases

The PV loop's four sides correspond to:

- A: Filling (volume increases, pressure low).
- B: Isovolumic contraction (volume constant, pressure increases).
- C: Ejection (volume decreases, pressure high).
- D: Isovolumic relaxation (volume constant, pressure decreases).

e. Flow Velocity Patterns

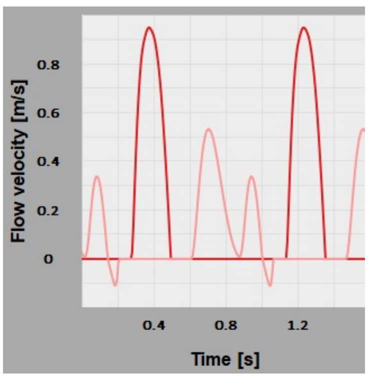
The difference in flow velocity patterns between the aortic and mitral valves is due to the distinct phases of the cardiac cycle during which these valves operate.

The **aortic valve** exhibits a **single-hump pattern** in its flow velocity curve. This is because the valve opens only during the *systolic phase* when the left ventricular pressure exceeds the aortic pressure, allowing blood to be rapidly ejected into the aorta. The resulting flow is unidirectional and sustained, producing one sharp peak that represents the entire ejection phase. Once ventricular pressure drops below aortic pressure, the valve closes, ending the flow.

In contrast, the **mitral valve** shows a **two-hump pattern**, corresponding to the two phases of *left ventricular diastolic filling*:

1. **E-wave (early passive filling):** Occurs immediately after the mitral valve opens. Blood flows passively from the left atrium to the left ventricle due to the pressure gradient, resulting in a prominent first peak.
2. **A-wave (active filling):** Occurs later in diastole due to *atrial contraction*, which pushes additional blood into the ventricle and creates a second, smaller peak in flow velocity.

Thus, the **aortic valve's single hump** reflects the *active ejection* of blood during systole, while the **mitral valve's double hump** reflects both *passive and active phases* of diastolic ventricular filling.

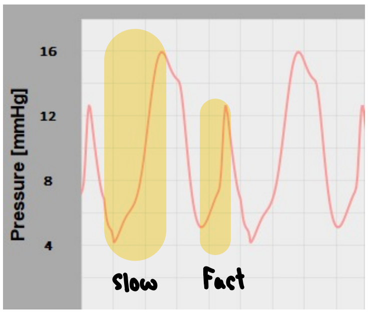


f. Analysis of Atrial Pressure Rises and ECG Correlation

Upon adjusting the y-axis scale of the atrial pressure graph in the CircAdapt simulator, two distinct increases in left atrial pressure can be identified during a single cardiac cycle:

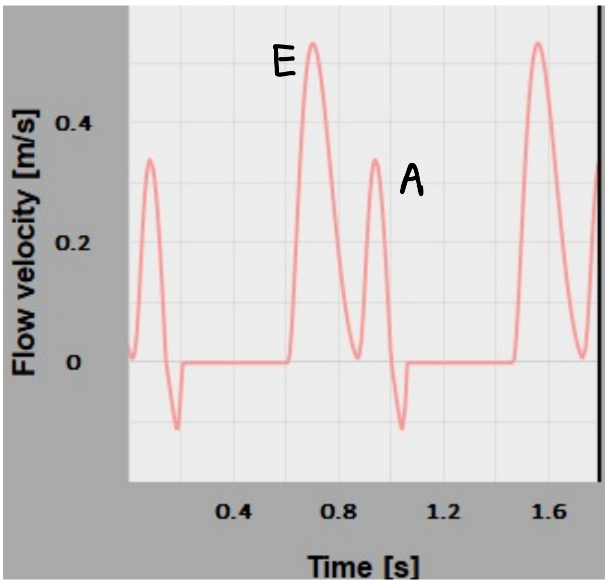
1. **Slow Rise in Atrial Pressure:** This gradual increase occurs during *ventricular systole*, when the mitral valve is closed. As the left ventricle contracts and ejects blood into the aorta, the mitral valve prevents backflow. Meanwhile, venous return from the pulmonary circulation continues, leading to a steady accumulation of blood in the left atrium. This causes a slow rise in atrial pressure, known as the **v-wave** in the atrial pressure curve.

2. **Sharp (Fast) Rise in Atrial Pressure:** This steeper increase happens just before ventricular systole and is caused by *atrial contraction*. As the atrium contracts to push additional blood into the ventricle, atrial pressure rises sharply. This contraction marks the final phase of diastolic filling and produces the **a-wave** in the atrial pressure curve.

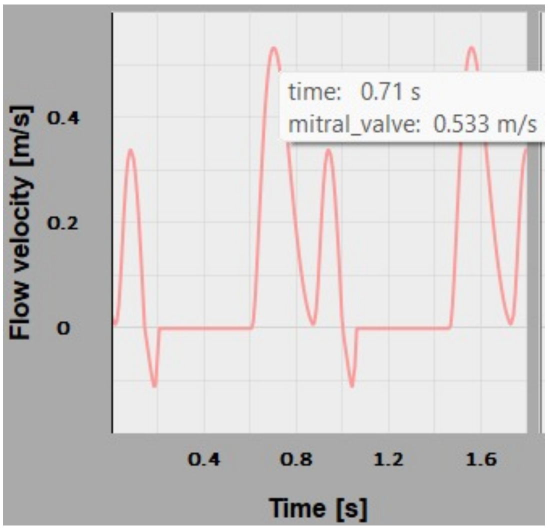


g. E and A Waves of Mitral Valve Flow

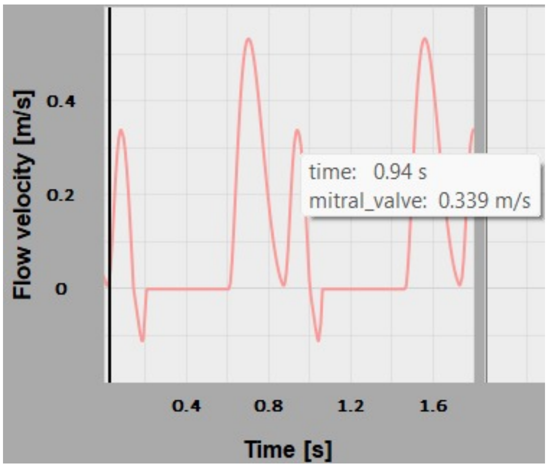
The mitral flow velocity signal shows the E-wave and A-wave.



h. E/A Ratio



(a)



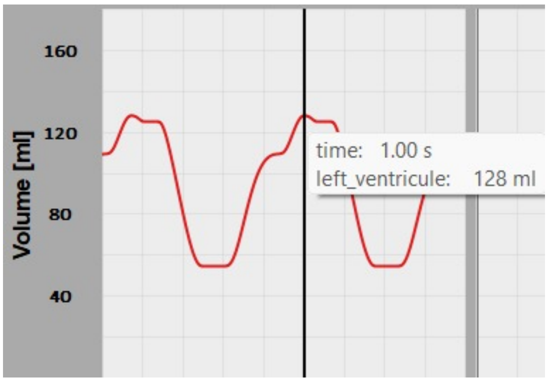
(b)

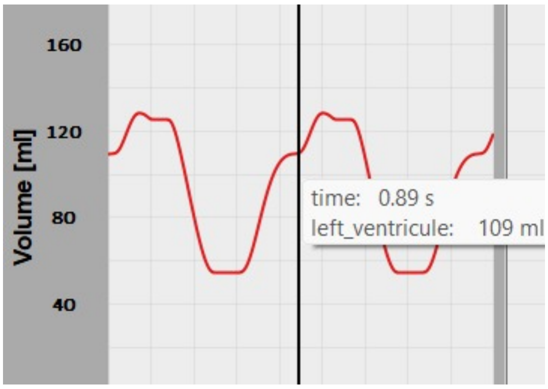
The E/A ratio is calculated as:

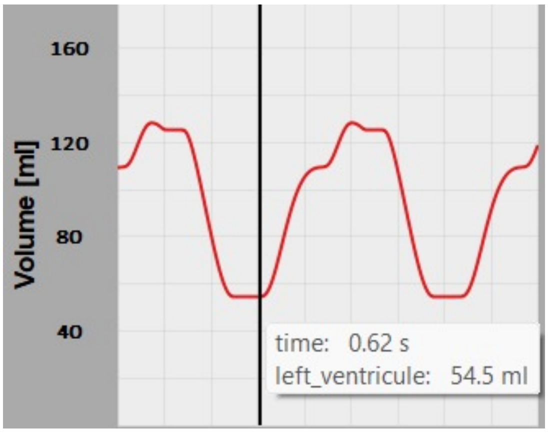
$$\mathrm{E/A} = \frac{0.533}{0.339} = 1.57$$

This indicates good diastolic function since it's larger than 1.

i. Passive vs. Active Filling







Due to passive filling = 109 ml - 54.5 ml = 54.5 ml
Due to active filling = 128 ml - 109 ml = 19 ml

j. Flow Rate Conversion

To convert blood flow velocity (V_{valve}) through a heart valve into volumetric flow rate (Q_{valve}), the essential anatomical property required is the **cross-sectional area** of the valve opening, commonly referred to as the *effective orifice area* (EOA).

$$Q_{\text{valve}} = A \times V_{\text{valve}}$$

Where:

- Q_{valve} is the volumetric flow rate (e.g., in m^3/s),
- A is the cross-sectional area of the valve (in m^2 or cm^2),
- V_{valve} is the blood flow velocity (in m/s).

The unit analysis also supports this relationship:

$$[\text{m}^2] \times [\text{m/s}] = [\text{m}^3/\text{s}]$$

Therefore, the **effective orifice area (EOA)** is the required anatomical property to convert flow velocity into flow rate.

2 Aortic Valve Stenosis

a. Preload and Afterload

Preload refers to the initial stretching of cardiac muscle fibers at the end of diastole, just before ventricular contraction. It is directly related to the *end-diastolic volume (EDV)*, which is the amount of blood filling the ventricles.

Afterload is the resistance the left ventricle must overcome to eject blood during systole. It is influenced by factors such as *aortic pressure* and *vascular resistance*.

b. Hemodynamic Changes

The procedure was done by increasing stenosis 5% at a time.

Stenosis	Peak velocity	Peak pressure	Peak volume
0%	1.25 ms^{-1}	121 $mmHg$	135 ml
5%	1.30 ms^{-1}	123 $mmHg$	135 ml
10%	1.37 ms^{-1}	123 $mmHg$	135 ml
15%	1.46 ms^{-1}	123 $mmHg$	135 ml
20%	1.49 ms^{-1}	124 $mmHg$	135 ml
25%	1.63 ms^{-1}	125 $mmHg$	135 ml
30%	1.73 ms^{-1}	126 $mmHg$	135 ml
35%	1.83 ms^{-1}	127 $mmHg$	135 ml
40%	1.96 ms^{-1}	129 $mmHg$	135 ml
45%	2.08 ms^{-1}	130 $mmHg$	136 ml
50%	2.28 ms^{-1}	132 $mmHg$	136 ml
55%	2.47 ms^{-1}	135 $mmHg$	136 ml
60%	2.69 ms^{-1}	139 $mmHg$	136 ml
65%	2.95 ms^{-1}	143 $mmHg$	137 ml
70%	3.28 ms^{-1}	149 $mmHg$	137 ml
75%	3.67 ms^{-1}	156 $mmHg$	139 ml
80%	4.18 ms^{-1}	171 $mmHg$	141 ml

Table 1: Relationship between stenosis and hemodynamic parameters

As shown in Table 1, **the velocity of the peak aortic valve increases** with stenosis. Based on:

$$Q_{\text{valve}} = A_{\text{valve}} \times V_{\text{valve}}$$

a reduction in valve area (A_{valve}) necessitates a rise in velocity (V_{valve}) to maintain constant flow.

Left ventricular pressure begins rising rapidly after 20% stenosis, while the **ventricular volume** increases more gradually. At 80% stenosis, the **maximum LV pressure reaches 171 mmHg** indicating increased afterload.

c. Effect of Aortic Stenosis on Preload, Afterload, and Cardiac Output

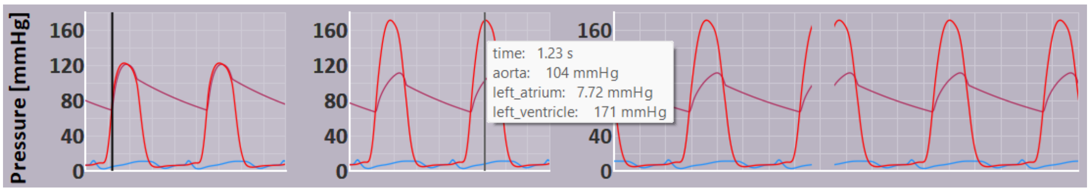
Aortic valve stenosis significantly affects both preload and afterload of the left ventricle:

Afterload increases markedly due to the narrowed valve opening, which presents greater resistance to blood flow during systole. The left ventricle must generate much higher pressures to overcome this increased resistance.

Preload may also rise over time, as incomplete ejection of blood leads to a higher end-systolic volume. Consequently, more blood accumulates during diastole, increasing the end-diastolic volume (EDV) and thus the preload.

These hemodynamic changes result in a decline in **cardiac output**, particularly as the condition progresses. While the heart may initially compensate through increased muscle mass (hypertrophy), prolonged elevated afterload impairs ventricular function, leading to reduced stroke volume and efficiency.

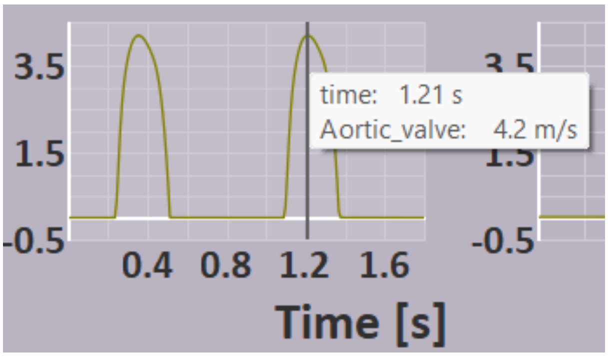
d. Pressure Drop



At maximal LV pressure (166 mmHg), aortic pressure is 115 mmHg. The pressure drop across the valve is:

$$\Delta p = 171 - 104 = 67\,\text{mmHg}$$

e. Bernoulli's Equation

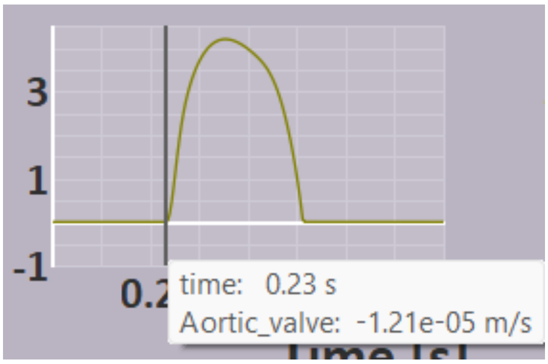


Using the simplified Bernoulli equation ($\Delta p = 4v^2$), with a maximal velocity of 4.2 m/s:

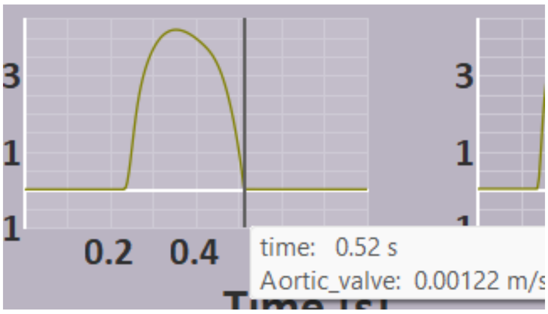
$$\Delta p = 4 \times (4.2)^2 = 70.56\,\text{mmHg}$$

This matches the 67 mmHg from part d.

f. Ejection Duration



(f)



(g)

Time taken to eject ventricular blood through the narrowed aortic valve is as follows,

$$\textit{Duration of ejection} = 0.52\,\mathrm{s} - 0.23\,\mathrm{s} = 0.29\,\mathrm{s}$$

g. External Pump Work

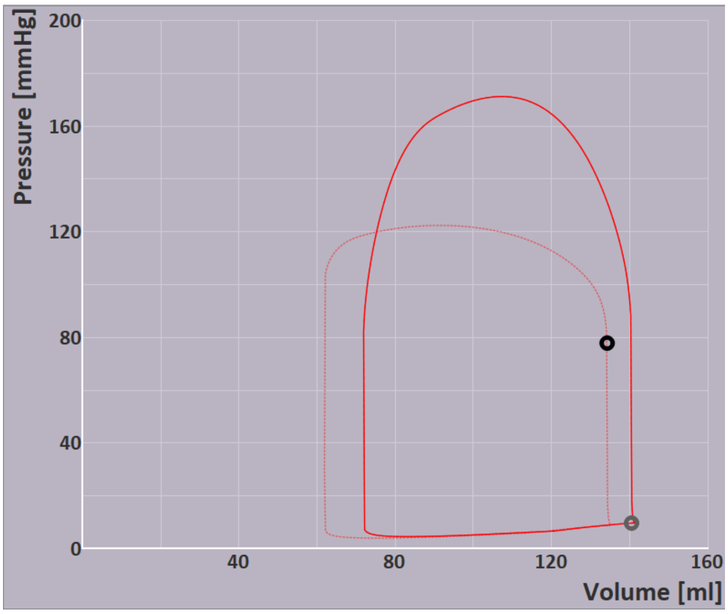


Figure 7: Screenshot of pressure -volume graph on the left ventricle.(dashed line-normal, solid line-AS)

External pump work in Normal person = 20 squares
External pump work at 80% stenosis = 24 squares
Increase in pump work = 4 squares

h. Myocardial Adaptation

To cope with the chronically increased pump work due to aortic stenosis, the left ventricular myocardium undergoes **concentric hypertrophy**. This thickening of the ventricular wall enables the heart to generate higher pressures and reduce wall stress.

While initially beneficial, the hypertrophied myocardium becomes less compliant, impairing diastolic filling. This leads to elevated diastolic pressure and a **further increase in afterload**, potentially contributing to diastolic heart failure over time.